these and other considerations. The photoactivation device **20** can apply light either without making direct contact with the skin or by making direct contact with the skin.

[**0072**] As FIG. **12** shows, once verteporfin is activated by light in the presence of oxygen, highly reactive, short-lived singlet oxygen and reactive oxygen radicals are generated. The singlet oxygen and reactive oxygen radicals cause local damage to inner wall or endothelium of the veins. Cells outside of contact with the activated verteporfin, however, are left unaffected.

[**0073**] Treatment by the system **10** and method just described intentionally causes injury to the inner vein walls. By controlling the clinically parameters above described (i.e., the dosage, delivery time and rate, operating conditions of the photoactivation device **20**, etc.,) the nature of the injury can be tightly controlled and localized.

[**0074**] The initial injury to the vein wall evokes a healing process (see FIG. **13**). During the healing process, the vein heals shut over time. The healing results in shrinkage of the spider vein, and eventually, complete obliteration of the spider veins in the targeted region, as FIG. **14** shows.

EXAMPLE

[**0075**] The superficial venous anatomy of the ears of New Zealand White Rabbits were treated by injecting light-reactive agent LS11 (Talaporfin Sodium) in doses selected to approximate a human dose, and thereafter exposing the superficial venous anatomy to light at a wavelength of 664 nm in dose periods ranging from 8 to 12 minutes. Visible alterations in the superficial venous anatomy due to shrinkage of veins in the treated regions were observed.

[**0076**] New Zealand Rabbits were chosen because of their large ears having easily identifiable superficial venous anatomy (as FIG. **19** shows). A total of twelve rabbits were treated. Each rabbit (weighing approximately six pounds) was intravaneously sedated for approximately 25 minutes using Ketamine and secured to a treatment table before being treated. The ears were shaved clean of hair and skin prepped with alcohol.

[**0077**] LS11 (Talaporfin Sodium, from Light Sciences Oncology, Inc.) was selected as the light reactive agent **14**. As FIG. **19** shows, the LS11 was administered intravenously by a syringe **18** and an IV administration line **68** into the superficial venous anatomy of one ear of each rabbit (the other ear serving as the Control). Different doses of LS11 (at a concentration of 0.125 mg per cc) were administered to different rabbits, at 0.25 mg/kg; 0.50 mg/kg; 1.0 mg/kg; and 1.5 mg/kg, respectively. The doses were selected to approximate a human dose of 3 mg/ml to 10 mg/ml (a dose of 0.2 to 0.5 mg/kg).

[**0078**] The doses were administered to each rabbit's ear intravenously in a slow bolus method over a period of 5 minutes. After a pre-selected delay of ten minutes following the injection, a light-applying carrier **60** of the type shown in FIG. **18**A (with a linear array of three LED's) was laid on the head of the rabbit over the treated ear, which was held tight against the carrier (see FIG. **20**). The light emitters **36** were operated at a wavelength of 664 nm. The power source was standard 110 V AC current.

[**0079**] For each LS11 dose, three different light doses—8 minutes, 10 minutes, and 12 minutes—were applied. Twelve rabbits were treated according to the protocol (four different LS11 doses at three different light doses).

[**0080**] At the conclusion of the light exposure, the treated ear was coated with aluminum oxide cream and a photograph taken.

[**0081**] FIG. **21** is a drawing based upon a representative photograph of a control ear (Control) and a treated ear (Treated) of one of the treated rabbits. FIG. **21** allows a side-by-side comparison between the superficial venous anatomy of a control ear and a treated ear (LS11 dose: 1.5 mg/kg; Light Dose: 8 min of 664 nm light). FIG. **21** demonstrates that treatment with the light-reactive agent LS11 (Talaporfin Sodium) in the manner described can serve to visibly alter the superficial venous anatomy, due to shrinkage of veins in the targeted region.

[**0082**] It should be appreciated that the devices, systems, methods, and protocols that have been described can provide minimally invasive, cost effective, and patient-friendly treatment of diseases or dysfunctions in all regions of the body that can be readily accessed by treatment agents carried by blood; e.g., cancers like breast and prostrate cancer; ear, nose, and throat conditions; periodontal disease; and diseases of the eye.

[**0083**] The foregoing is considered as illustrative only of the principles of the invention. Furthermore, since numerous modifications and changes will readily occur to those skilled in the art, it is not desired to limit the invention to the exact construction and operation shown and described. While the preferred embodiment has been described, the details may be changed without departing from the invention, which is defined by the claims.

I/we claim:

1. A method for treating a spider vein comprising

providing a reactive agent that is controllably activated by the application of a prescribed form of energy,

distributing the reactive agent at, in, or near an inner wall of a spider vein, and activating the reactive agent by applying the prescribed form of energy that activates the reactive agent in situ to cause localized injury to the inner wall of the spider vein.

2. A method according to claim 1

wherein the prescribed form of energy comprises electromagnetic radiation.

3. A method according to claim 1

wherein the prescribed form of energy comprises light energy.

4. A method for treating a spider vein comprising

providing a light-reactive agent,

distributing the light-reactive agent at, in, or near an inner wall of a spider vein, and

activating the light-reactive agent by applying light energy having a wavelength that activates the light-reactive agent to cause localized injury to the inner wall of the spider vein.

5. A method according to claim 4

wherein the light-reactive agent comprises verteporfin.